

Necrosis of Falciform Ligament Complicating a Cholecystitis with a Pylephlebitis and Abscess of the Abdominal Wall

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Abstract

Case Report

Gangrene of the hepatic ligaments (falciform and/or round) is an exceptional and poorly understood condition and may present a diagnostic dilemma. We report a case of necrotic falciform ligament abscess with pylephlebitis secondary to pycholecyst. The clinical picture was impressive and noisy. The diagnosis was evoked on the CT scan, with the discovery of a circumscribed lesion of fatty density, pre-hepatic, in the fissure of the round ligament, at the junction of segments III and IV of the liver. The interest of surgical exploration is mainly diagnostic insofar as the evolution is always favourable, independently of the modalities of excision of the necrotic zones. In this article, we discuss the diagnostic and therapeutic features of this entit.

Keywords: Necrosis of falciform ligament, cholecystitis, pylephlybitis.

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INTRODUCTION

The term “Intra-Abdominal Focal Fat Infarction” (IFFI) encompasses a range of conditions where infarction of fatty tissue is the underlying pathological process, including Epiploic appendagits, omental infarction, fat necrosis related to trauma or pancreatitis as well as rarer entities such as falciform ligament infarction.

Reviews of the English and French literature have identified only a few cases. Preoperative diagnosis is difficult.

Rarely complications such as liquefaction and secondary abscess formation may arise with associated deterioration in symptoms and signs prompting a change in management including Laparoscopic resection or Percutaneous drainage.

Pylephlebitis (septic portal thrombosis) is an infectious phlebitis of the portal venous system, most often resulting from an extension of suppurated thrombophlebitis secondary to an intra-abdominal or pelvic infectious focus with a major morbi-mortality.

We present a case of a patient with Necrosis of falciform ligament complicating a cholecystitis with pylephlybitis and abscess wall formation.

PATIENT CASE

The patient was a 72-year-old woman with hypertension who had been poorly monitored and consulted for abdominal pain for 5 days.

The examination revealed a fever of 38.8°C and tenderness in the supra-umbilical region, accentuated in the epigastrium and right hypochondrium.

There was a hyperleukocytosis of 19,780 elements/mm³, a CRP of 230 mg/l, a normal lipase of 75 IU/L and a normal transaminase level (ALT of 50 IU/L and ASAT of 39 IU/L).

The abdominal X-ray was normal.

Abdominal ultrasound showed a heterogeneous hyper-echoic area located to the right of the midline, a thickened and laminated lithiasis gallbladder with a diffuse peritoneal effusion.

A CT scan was performed urgently and showed a swollen appearance with poorly enhanced edematous infiltration of the falciform ligament extending to the hepatic hilum (Figure 1 and 2), in the left subhepatic area down to the anterior abdominal wall in connection with necrosis of the falciform ligament

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with a right paralumbilical intraperitoneal collection (Figure 3) complicated by a left pylephlebitis (septic left portal thrombosis) (Figure 4), associated with a

peritoneal effusion The gallbladder was in lithiasis pyocholecyst while the intra and extra hepatic bile ducts were of normal caliber.

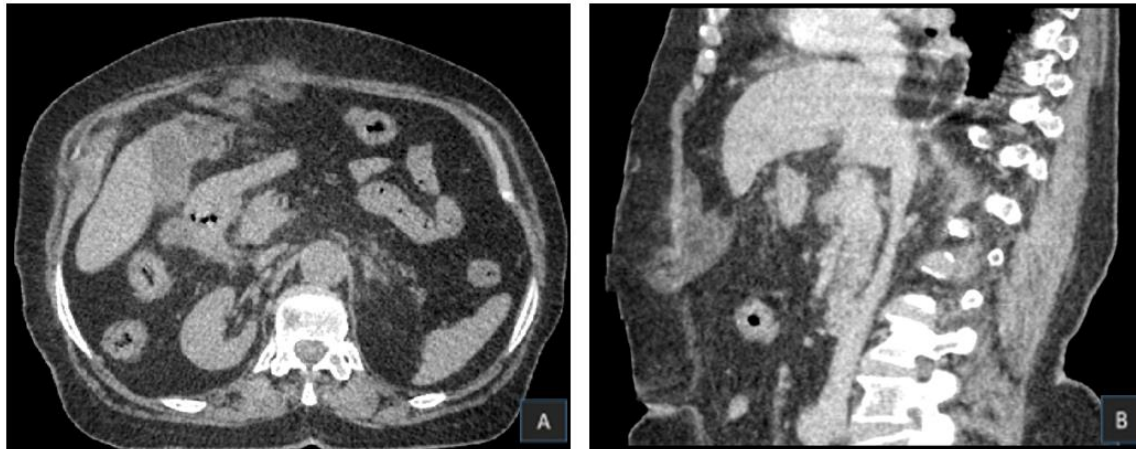


Figure 1: Swollen and infiltrated appearance of the falciform ligament, poorly enhanced by PDC, Extensive infiltration of the liver hilum and anterior abdominal wall A: axial view B: sagittal view

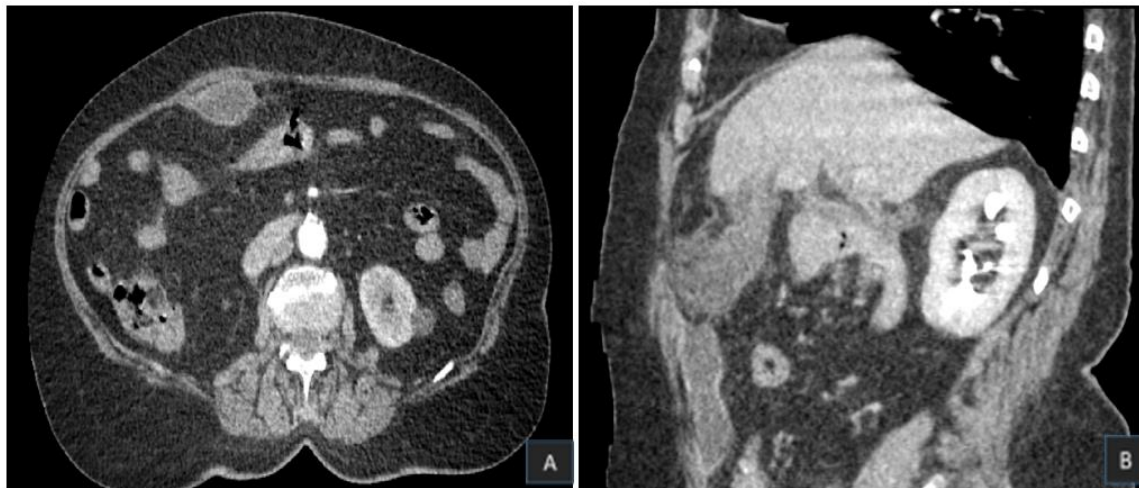


Figure 2: Right umbilical collection, spontaneously hypodense, peripherally enhanced by PDC axial view (A) sagittal view (B)

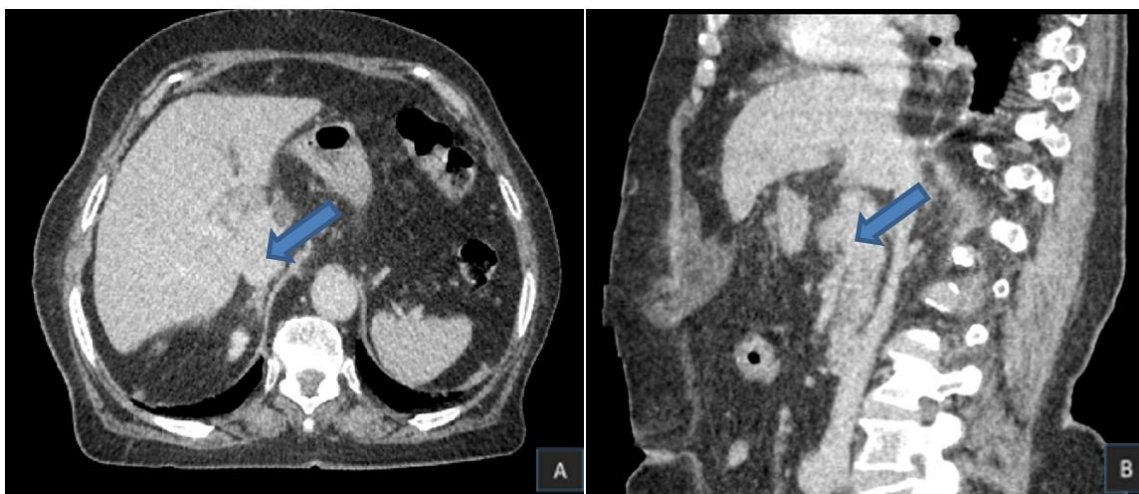


Figure 3: Thrombosis of the left portal branch (red arrow); axial view (A) sagittal view (B)

The patient was taken to the operating theatre and underwent emergency surgery. Surgical exploration, by median laparotomy, revealed a subhepatic and epigastric shielding made of a pyocholecystic gallbladder associated with an abscess with necrosis of the round and sickle ligaments of the liver which extended to the hepatic sinus between segments III and IV of the liver (Figure 4) and in the periumbilical area with purulent collections in the right and left parietocolic gutters and in the inter-apes.

The procedure consisted of aspiration of the purulent collections, removal of all necrotic tissue from the falciform and round ligaments, and antegrade cholecystectomy.

The abdominal cavity was well washed and drainage was ensured by three Redon drains placed respectively in the right subphrenic, subhepatic and at the level of Douglas and by a Delbet blade in the periumbilical region.

The patient was put on a curative dose of anticoagulant treatment with broad-spectrum antibiotic therapy and the postoperative course was simple.

Anatomical examination of the resected ligament fragments revealed inflammatory and necrotic changes.



Figure 4: Pictures of intraoperative necrotic and abscessed aspect of the falciform ligament

DISCUSSION

The Falciform ligament is a structure arising at the umbilicus and continues onto the anterior aspect of liver in continuity with the umbilical fissure. It is a double fold of peritoneum that anatomically divides the liver into the right and left lobes and extends from the superior edge of the liver to the inferior border of the diaphragm.

The falciform ligament contains the ligamentum teres within the lower edge, paraumbilical veins, and variable amount of extra peritoneal fat. The ligamentum teres is a remnant of the obliterated umbilical vein [1].

Pathologies such as cystic lesions, internal ligament hernias, lipomas, hematomas, inflammation and necrosis of the ligaments have been reported but are very rare [2, 3].

The arterial supply of the falciform ligament originates from a thin branch of the right hepatic artery, which is anastomosed to the superficial inferior epigastric artery. Venous drainage flows directly into the paraumbilical vein and portal vein. Falciform ligament necrosis may likely develop if the embolization of this artery or venous thrombosis occurs [4].

The falciform ligament can be secondarily affected by surrounding inflammation, however primary

pathologies are rare. Primary infarction of the ligament is rare with very few cases reported in the literature.

A septic or thrombotic cause as well as torsion could lead to gangrene of the falciform ligament [6, 7]. Necrosis of the falciform ligament is a rare cause of acute abdominal pain and may mimic the clinical picture of cholecystitis, perforated duodenal ulcer or pancreatitis [8, 9].

The pathophysiology of necrosis of the falciform and round ligaments remains unclear. Two theories have been put forward. The first suspects an infectious bacterial origin by diffusion of germs of the first one is due to a digestive tract infection through an umbilical vein that remained partially permeable [7-11]. The second suggests an ischemic origin with a mechanism similar to that of appendicitis [8-11]. In our case, we hypothesize that pycholecyst and secondary portal pyaemia and routing through the para-umbilical veins may cause infectious seeding of the falciform and/or round ligaments, resulting in abscess formation and/or necrosis.

Pylephlebitis (septic portal thrombosis) is an infectious phlebitis of the portal venous system, most often resulting from an extension of suppurated thrombophlebitis secondary to an intra-abdominal or pelvic infectious focus with a major morbi-mortality. The most frequently found germs are *Escherichia coli* and *Streptococcus* [12]. Idiopathic pylephlebitis is described in 1-6% of cases [13].

Preoperative diagnosis is difficult. This is due to a lack of knowledge of the pathology and an unsympathetic clinic.

At sonography it appears as an oval echogenic, non-compressible mass at the point of maximal tenderness [14].

Sonography may be useful to demonstrate that the lesion does not move with breathing, due its superficial extra peritoneal nature [15, 16]. Blood flow is typically absent on color Doppler unlike other inflammatory conditions.

The most common CT feature is an oval area of heterogeneous fat attenuation surrounded by a ring of soft tissue (hyper attenuating rim sign) that represents adjacent inflamed peritoneum, sometimes with a central area of high attenuation (central dot sign) due to venous thrombosis, associated to adjacent inflammatory changes [17].

However, rarely fat necrosis can undergo complications such as liquefaction or secondary abscess formation as demonstrated in our case series which necessitates a change in the management in the form of percutaneous drainage or laparoscopic resection.

The presence of thick enhancing walls, fluid levels and air within the lesion heralds the formation of an abscess.

Uncomplicated IFFI is usually self-limiting and managed conservatively with oral antinflammatory therapy [18].

Treatment is usually removal of the ligament because of symptomatic relief and diagnostic uncertainty, although there are few reports of cases treated conservatively. Increasing radiologist awareness of sickle ligament necrosis imaging will allow this rare entity to be included in the differential diagnosis of the acute abdomen. Laparoscopy could be a good diagnostic and therapeutic alternative [19].

CONCLUSION

Isolated necrosis of the round ligament of the liver is a rare cause of acute abdomen. Its diagnosis remains difficult despite the contribution of abdominal CT. The evolution has always been favorable. The interest of surgery has been mainly diagnostic. In case of doubt, an exploration with or without resection of the round ligament, under laparoscopic video control, seems to be an alternative adapted to the benignity of this pathology.

ADDITIONAL INFORMATION

Disclosures

Human Subjects: Consent was obtained by all participants in this study.

Conflicts of Interest

In compliance with the ICMJE uniform disclosure form, all authors declare the following:

- **Payment/Services Info:** All authors have declared that no financial support was received from any organization for the submitted work.
- **Financial Relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work.
- **Other Relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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